

ORAL

DERMATOLOGY



Important Oral Questions & Answers

سؤال و جواب

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PSORIASIS

Q- What is the Primary & Secondary Lesion of Psoriasis ?

Primary Lesion: Red (Erythematous) Plaque.

Secondary Lesion: White Silvery Scales.

Q- What are the Differential Diagnosis of Koebner Phenomenon ?

1- Psoriasis, 2- Lichen Planus, 3- Vitiligo, 4- Wart, 5- Molluscum Contagiosum.

Q- What are the Types of Psoriasis ?

1- Psoriasis Vulgaris, 2- Scalp Psoriasis, 3- Nail Psoriasis, 4- Guttate Psoriasis,
5- Napkin Psoriasis, 6- Psoriatic Arthritis, 7- Psoriatic Inverses, 8- Palmo-Planter
Psoriasis, 9- Erythrodermic Psoriasis.

Q- What are the Danger Types of Psoriasis ?

1- Erythrodermic Psoriasis. 2- Palmo-Planter Psoriasis.
3- Psoriatic Arthritis (Arthropatic Psoriasis).

Q- What are the Sites that Should be Examined in Each case of Psoriasis ?

Extensor Body Surfaces (Knee & Elbow joints), Scalp, Nails, Trunk,
Also Flexor Surfaces, Sacral Area.

Q- What are the Nail Changes of Psoriasis ?

1- Pitting in Nail Plate, 2- Subungual Hyperkeratosis, 3- Onycholysis, 4- Oil Drop Sign.

Q- What are the Local Treatment of Psoriasis ?

Emollients (Vaseline), Keratolytic Agent (Salicylic Acid 5%), Coal Tar, Dithranol (or Anthraline), Vit D3 Analogus, Local Retinoid, Topical Steroid or Tacrolimus, Topical UVB Ray (Broad Bond → 290-320 nm , or Narrow Bond → 311-319 nm "Best").

Q- What are the Systemic Treatment of Psoriasis ?

- 1- **PUVA**; "**Psoraline + UVA**": interfering with DNA Synthesis.
- 2- **Retinoid**; "**Vit A Derivatives** ": Necessary for Normal Keratinization.
- 3- **Methotrexate**; Drug Of Choice for Psoriatic Arthritis Once Per Week **10-25mg**, Given with **Folic Acid** (For; Megaloblastic Anemia).
- 4- **Cyclosporine A**; Immunosuppressant, inhibits Production of Interleukin-2.

Q- What are the Investigations that has to be Done Before & During Giving of Systemic Treatment ?

1. **Lipid Profile**: for HyperLipidemia Due to **Retinoid**.
2. **Liver Function Test (LFT)**: for Hepato-toxicity Due to **PUVA & Methotrexate**.
3. **Renal Function Test (RFT)**: For Nephro-toxicity Due to **Cyclosporine**.
4. **Complete Blood Count (CBC)**: For Bone Marrow Suppression Due to **Methotrexate**.
Rare → **Bone Marrow Biopsy**: for Bone Marrow Suppression Due to **Methotrexate**.

Q- What are the Side Effect of Systemic Retinoid ?

- 1- Teratogenic (So you Should Stop the drug **2** years, and then Get Pregnant).
- 2- Hyperlipidemia (So; Do **Lipid Profile**).
- 3- Increase Liver Enzymes (So; Do **LFT**).
- 4- Bone Marrow Suppression (So; Do **CBC**).
- 5- Dryness of the Skin & Hair loss.

Q- What are the Wave Length of UVA & UVB that Used in Treatment of Psoriasis ?

UVA: **320 - 400 nm**.

UVB: Broad bond UVB → **290 - 320 nm**, Narrow bond → **311 - 319 nm** (Best).

Q- What are the Manifestations of Erythrodermic Psoriasis ?

- 1- Disturbance in Water and Electrolytes Balance.
- 2- High Cardiac Output (Due to **increase** Blood Supply of the **Dermis**).
- 3- Hypo-Proteinemia (Due to **Extensive Loss** of Keratin **Protein**).
- 4- Sever Hypothermia.
- 5- V.D → Hypotension → **Shock**.

VESICULO-BULLOUS DISORDERS

Q- What are the Vesiculo-Bullous Diseases ?

Group of Disorders Characterized by **Bullous** “Blister” and/or **Vesicle** formation.

Q- Enumerate the Auto-Immune Bullous Diseases ?

- 1- Pemphigus (**Intra-Epidermal**).
- 2- Haily Haily (**Intra-Epidermal**).
- 3- Pemphigoid (**Sub-Epidermal**).
- 4- Dermatitis Herpetiformis (**Sub-Epidermal**).

Q- What are the Difference Between Pemphigus & Pemphigoid ?

Pemphigus Vulgaris	Bullous Pemphigoid
The Bullae are: Life Threatening, Non Pruritic, Painful , Flaccid , Not Filled with Blood, Easily Rupture.	The Bullae are: Not Life Threatening, Pruritic, Mild Painless , Tense , Filled with Blood Difficult to Rupture.
Positive Nikolsky Sign.	Negative Nikolsky Sign.
Pathology in Desmosomes .	Pathology in Hemi-desmosomes .
50-70% Has Oral Lesion	10-15% Has Oral Lesion.
40 – 60 Years Old.	60 – 80 Years Old “ Elderly ”.
Very Bad Condition.	Good General Condition.
1. Biopsy Shows → Intra-Epidermal Bullous. 2. Direct Immuno-fluorescence Study, Shows→ Inter Cellular Deposition of IgG . 3. Tzank Smear is Positive .	1. Biopsy Shows → Sub-Epidermal Bullous. 2. Direct Immuno-fluorescence Study, Shows→ Deposition of IgG on Basement Membrane . 3. Tzank Smear is Negative .
Treated With Large Dose Systemic Steroid.	Treated With Small Dose Systemic Steroid.

Q- What Does Direct Immunofluorescence Show in Pemphigus & Pemphigoid ?

- **Pemphigus**; Direct Immuno-fluorescence Study Shows → Inter Cellular Deposition of IgG.
- **Pemphigoid**; Direct Immuno-fluorescence Study Shows → IgG Deposition on Basement Mem.

Q- What are the Differential Diagnosis of Mouth Ulcer ?

Pemphigus, Pemphigoid, Lichen Planus, Steven Johnson Syndrome, Herpes Simplex, Syphilis, Bahcet Disease, SLE, Aphthous Ulcer.

Q- What are the Symptoms & Signs of Dermatitis Herpetiformis ?

Intensely Pruritic, Painless (But Burning), Grouped Vesicles of Symmetrical Distribution, Associated with Celiac Disease (Gluten Sensitivity).

Q- What Does Direct Immunofluorescence Shows in Dermatitis Herpetiformis ?

Direct Immuno-fluorescence Study Shows → Deposition of IgA.

Q- What is the Drug that Used in Treatment of Dermatitis Herpetiformis ?

Dapsone.

Q- What are the Side Effect of the Drug that Used in Treatment of Dermatitis Herpetiformis ?

1. **Pancytopenia**; "Haemolytic Anemia (Especially in pt with G.6.P Deficiency) + Leukopenia + Thrombocytopenia".
2. **Peripheral Neuropathy**.
3. **Hepatitis**.

Q- Mention One Disease Treated By Same Drug that Used in Dermatitis Herpetiformis ?

Leprosy.

VITILIGO & ALBENISM

Q- Define Vitiligo ?

It is an Acquired De-Pigmented Cutaneous Disorder Caused by Loss of Melanocyte.

Q- What are the Pathogenic Theories of Vitiligo ?

- 1- **Auto immune Theory**; (Commonest) Presence of "**Ab**" against Melanocyte, associated with Other Auto immune Diseases, (Ex; DM Type1, Addison Disease, Hypothyroidism).
- 2- **Neurotoxic Theory**; Present in Certain Dermatomes neurotoxins Destruct Melanocyte.
- 3- **Self Destruction**; (Apoptosis).
- 4- **Stress**.

Q- What are the Investigations of Vitiligo ?

Woods Lamp → Milky White.

Blood Investigations; **CBC** → High Lymphocytes.

Q- How to Treat Vitiligo ?

1. Reassurance.
2. Protect the Patient from Excessive Sun Exposure by Sun Screen, Then According to Type:

Localized Type:	Multiple diffuse Type:
1. Local Steroids. 2. Macrolide; Ex→ Tacrolimus. 3. Cosmetic Camouflage; Cream Cover Color of the Skin "Especially in Segmental Type".	1. PUVA (Psoraline + UVA). 2. Systemic Steroids → Used in <u>Unstable Active</u> Vitiligo. 3. Narrow bond UVB (290-300nm) → Good in <u>Non active</u> Vitiligo. 4. Melanocyte Graft.

- If Patient had **90%** of his body affected with Vitiligo; → Then Do "De-Pigmentation" by Ethyl Ether Hydroquinone.

Q- What is the Color of Woods Lamp in Vitiligo ?

Milky White.

Q- Define Albinism ?

Congenital Autosomal Recessive Disease Characterized by:
Generalized Hypo-Pigmentation Due to Loss of Melanin Pigment Due to Deficiency of Tyrosinase Enzyme, Affect Skin, CNS, Eyes.

Q- How to Differentiate Between Vitiligo & Albinism ?

Albinism	Vitiligo
Congenital Autosomal Recessive.	Acquired.
Decrease of Tyrosinase Enzyme.	Unknown Melanocyte Destruction .
Hypo -Pigmented areas.	De -Pigmented areas.
Affect Skin, CNS. Eyes.	Affect Skin Only .
C\P : White Hair, Iris, Nystagmus, Skin Malignancy	No
Loss of Melanin Pigment .	Loss of Melanocytes .
Generalized.	Local.

ERYTHEMA

Q- Enumerate Types of Erythema ?

- 1- Erythema Nodosum.
- 2- Erythema Multiform.
- 3- Erythema Induratum.
- 4- Palmo-Planter Erythema.
- 5- Erythema Annulare Centrifugium (**EAC**).
- 6- Erythema Infectiosum (**Fifth Disease**).
7. Erythema Marginatum (**Rheumatic Fever**)
- 8- Sweet Syndrome (**Acute Febrile Neutrophilic Dermatositis**).
9. Drug Induced Erythema.

Q- What are the Causes of Erythema Nodosum ?

It has Poly Athiology (Many Causes):

1. Infections:

- Bacterial; Streptococci (**Most Common**), TB, Leprosy, Mycoplasma, Salmonella, Shigella.
- Fungal; Histoplasmosis.
- Protozoal; Toxoplasmosis.

2. **Drugs**; OCP, Sulphonamides, Gold.

3. **Disease**; Sarcoidosis, Crhons Disease (**IBD**), Bahcet Disease.

4. **Pregnancy**.

5. **Malignancy**.

6. **Idiopathic**.

Q- What are the Differential Diagnosis of Erythema Nodosum ?

- 1- Erythema Induratum.
- 2- Cellulitis.
- 3- Phlebitis.
- 4- Insect bite.

Q- What are the Treatment of Erythema Nodosum ?

- 1- Bed Rest & Elevation of Legs
- 2- Cold Compression & NSAID (Indomethacin, Ibuprofen).

Q- What is the Special Lesion of Erythema Multiform ?

Target Lesion (**Iris Lesion**).

Q-What are the Types of Erythema Multiform & Which One is Worst Prognosis ?

Types of Erythema Multiform:

- 1- Erythema Multiform Minor.
- 2- Erythema Multiform Major (**Steven Johnson Syndrome**) → Worst Prognosis.

Q- How to Treat Erythema Multiform ?

According to Type:

Minor Type → Topical Steroid.

Major Type → Systemic Steroid + Systemic Anti-Biotic.

DERMATITIS (ECZEMA) & URTICARIA

Q- What are the Classifications of Dermatitis (Eczema) ?

1- Exogenous Eczema (Dermatitis):

- Primary Irritant Dermatitis.
- Contact Allergic Dermatitis.
- Photo-Dermatitis.

2- Endogenous Eczema (Dermatitis):

- Atopic Dermatitis.
- Seborrhoeic Dermatitis.
- Discoid (**Annular**) Eczema.
- Stasis Dermatitis (**Venous Ulcer**).
- Asteatotic Dermatitis (**Eczema Craquele**).
- Pompholyx.
- Neuro Dermatitis (**Lichen Simplex Chronicus**).

Q-How to Differentiate Between Atopic Dermatitis & Seborrhoeic Dermatitis ?

Atopic Dermatitis	Seborrhoeic Dermatitis
- Unknown Cause but Mainly Genetic - Has Positive Family History. - Associated with Other Atopic Diseases. - Positive Patch Test. - Occurs at Any Site. - According to Age Classified into Infantile, Childhood, & Adult Type.	- Occurs Due to Androgen Control. - Has No Family History. - Not Associated with Atopic Diseases. - Negative Patch Test. - Occurs in Sites of Sebaceous Glands in All Body Except; Palms, Soles, Lips. - According to Age Classified into Infantile & Adult Type.

Q- What are the Criteria of Atopic Dermatitis ?

1. Loss of Outer $\frac{1}{3}$ of Eye Brows.
2. Thickening of Lower Eye Lid.
3. Extra Fold around Eye (**Demorgan Sign**).
4. Dirty Neck Sign.
5. Waisting of Thener & Hypothener Muscles.
6. Palmer Creases More Visible (**Thick**).
7. Lichnification & Scratch Marks.

Q- What are the Side Effects of Local & Systemic Steroids ?

Local Steroid Side Effect:	Systemic Steroid Side Effect:								
1. Skin Atrophy. 2. Stria. 3. Telangectasia. 4. Hypo & Hyper-Pigmentation. 5. Decrease Local Immunity → Risk of Infection.	<u>Cushing Syndrome</u> Due to <u>Prolonged</u> Use of Systemic Steroid: <table> <tr> <td>1. Cataract.</td><td>2. Glaucoma.</td></tr> <tr> <td>3. DM.</td><td>4. Hypertension.</td></tr> <tr> <td>5. Obesity.</td><td>6. Osteoporosis.</td></tr> <tr> <td>7. Hirsutism.</td><td>8. Acne.</td></tr> </table>	1. Cataract.	2. Glaucoma.	3. DM.	4. Hypertension.	5. Obesity.	6. Osteoporosis.	7. Hirsutism.	8. Acne.
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7. Hirsutism.	8. Acne.								

Q- What are the Classification of Anti-Histamine Drugs ?

H1 Antagonist	H2 Antagonist
- <u>First Generation (Sedative)</u>: Cause <u>Sedation & Sleep</u> Because They <u>Cross</u> Blood Brain Barrier. They <u>Includes</u> → Promethazine (<i>Phenergan</i>), Hydroxyzine, Diphenhydramine. - <u>Second Generation (Non-Sedative)</u>: <u>Doesn't</u> Cause Sedation & Sleep Because They <u>Can't</u> Cross Blood Brain Barrier. They <u>Includes</u> → Loratidine, Disloratidine, Terfenadine, Azelastine, Acrivastine, Cetirizine. - <u>Third Generation</u>: <u>Includes</u> → Fexofenadine, Lerocetirizine.	It Includes: <u>Cimitidine, Ranitidine, Nizatidine.</u> Act in <u>Stomach</u> → <u>Decrease HCL</u> .

Q- What are the Urticaria Triade ?

1- Erythema 2- Edema 3- Itching.

Q- Enumerate Types of Physical Urticaria ?

- 1- Aquagenic Urticaria: Related to Polycythemia Rubra Vira.
- 2- Solar Urticaria: Related to Sun Light.
- 3- Colar Urticaria: in Nose & Tip of Fingers.
- 4- Pressure Urticaria.
- 5- Vibration Urticaria.
- 6- Dermographism Urticaria (Skin Drawing).
- 7- Cholinergic Urticaria (Smokers).

ACNE & ROSACEA

Q- What are the Aithiology of Acne ?

- 1- Seborrhoic Activity: (No Acne between 2-6 Years; Because No Activity of Sebaceous Gland).
- 2- Genetic: Hereditary, Common in White Men.
- 3- Hormonal: Excess Androgen.
- 4- Bacterial: Staphylococcus Epidermidis, Coryneabacteria
- 5- Psychological: Stress increase Androgen.
- 6- Mechanical: By Occlusion Pressure on Skin (**Ex**: Cosmetics).
- 7- **Drugs**: Anti TB, Anti Epilepsy, Steroid, Sedative, Lithium.

Q- Mention the Primary Lesions of Acne ?

Comedone, Papules, Pustules, Nodules, Cyst.

Q- What are the Types of Acne ?

1. Acne Vulgaris (Commonest Type).
2. Infantile Acne.
3. Cosmetic Acne.
4. Excoriates Acne.
5. Drug Induced Acne + Steroid Induced Acne.
6. Acne Conglobata (Sever Type).
7. Acne Fulminans. "Acute Febrile Ulcerative Acne" (Sever Type).

Q- What are the Drugs that Used for Treatment of Acne ?

Local Treatment:	Systemic Treatment:
1. Local <u>Anti-Biotic</u> : Erythromycin or Clindamycin at Day, or Combined with Benzyl Peroxide at Night.	1. Systemic <u>Anti-Biotic</u> : - <u>Tetracycline</u> → Decrease Free Fatty acid in Sebum. Dose → 500-1000mg\Day\6 Weeks (on Empty Stomach). C\I in Children; Because <u>Irreversible</u> Teeth Colorization. - <u>Erythromycin</u> is D.O.C in Pregnancy. - If <u>Azithromycin</u> is Used; you Need to Give it for 3 Days Then Stop it For 10 Days , Then Use for 3 Then Stop 10 .
2. Local <u>Retinoid</u> (Vit A Derivatives) : Act as <u>Comedonolytic</u> .	2. Systemic <u>Retinoid</u> : <u>Decrease Activity</u> of Sebaceous Gland Ex; Isotretinon, (Dose: 0.5-1mg\kg\12-16 Weeks).
3. Benzyl Peroxide : → <u>Anti-Bacterial</u> Also <u>Comedonolytic</u>	3. Intra-Lesion Steroid : For Deep Nodulo-Cyst.
4. Azelaic acid : 20% Decrease <u>Hyper-</u> <u>Pigmentation</u> .	4. Chemical Peeling : with Glycolic acid.

Q- Define Rosacea ?

Chronic Inflammatory **Acne** form Disorder of **Pilosebaceous Follicle** Characterized by: Sebaceous gland **Hyperplasia**, **Normal** Sebum Production, **Telangiectasia** (Dilated Dermal Blood Vessels), But **NO Comedone**.

Q- What are the Difference Between Acne & Rosacea ?

Rosacea	Acne
Limited in Face	Face, Chest, Shoulder
Telangiectasia	No Telangiectasia
No Comedone	Comedone
Normal Sebum Production	High Sebum Production
Age Between 30-50 Years Old	Age Between 12-20 Years Old
Female > Male	Male > Female

Q- What are the Complications of Rosacea ?

Blephritis, Conjunctivitis, Keratitis, Episcleritis,
Lymph-Edema around Eyes, Over-Growth of Soft Tissue (Nose → **Rhinophyma**).

Q- What is Rhinophyma ?

Over Growth of Soft Tissue of the Nose (**Enlargement of Nose**).

Q- What are the Treatment of Rhinophyma ?

Surgical.

HAIR DISORDERS

Q- Define: Alopecia Totalis, Alopecia Universalis, Hirsutism, Hypertrichosis ?

Alopecia Totalis: Complete Loss of Scalp Hair.

Alopecia Universalis: Loss of Hair All Over the Body.

Hirsutism: Excessive Growth of Thick Dark Hair in Location Where Hair growth in Female Usually absent or Minimal (in Face, Chest, Areola).

Hyper-trichosis: Excessive Terminal Hair Growth in Non Androgenic Distribution.

Q- What are the Differential Diagnosis of Scarring & Non Scarring Alopecia ?

Non Scarring	Scarring (Scatrical)
1- Alopecia Areata. 2- Tractional Alopecia. 3- Hair Pulling Habit. 4- Trichotillomania. 5- Male Pattern Alopecia. 6- Non Inflammatory Tinea Capitis. 7- Scalp Psoriasis	1- Inflammation: Lichen Planus, Bullous Pemphigoid, Discoid Lupus, Scleroderma, Sarcoidosis. 2- Infection: - <u>Bacterial</u> → Furuncle, Carbuncle. - <u>Fungal</u> → Kerion, Favus. - <u>Viral</u> → Herpes Zoster. 3- Genetic: Lamellar Ichthyosis. 4- Physical: Burns, Trauma, Chemical. 5- Malignancy.

Q- What are the Stages of Male Pattern Alopecia ?

- 1- Frontal Thinning.
- 2- Frontal & Crown Balding.
- 3- Extensive Balding.
- 4- Sever Balding.

Q- What are the Treatment of Male Pattern Alopecia ?

Symptomatic Treatment → Topical Minoxidil Lotion.

Q- Mention Seven Causes of Diffuse Hair Loss ?

Endocrine Disorders: Hyperthyroidism, Hypothyroidism, Hypoparathyroidism, Hypopituitarism.

Drugs: Chemotherapy, Immuno-Suppressant Drugs (**Cyclosporine**), Cytotoxic Drugs (**Cyclophosphamide**), Retinoid (**Excess Vit A**), Anti-Coagulant (**Heparin**), Colchicine.

Diseases: SLE, Malnutrition, Stress.

GENODERMATOSIS

Q- Define Genodermatosis ?

They are Group of Inherited Genetic Skin Conditions.

Q- Enumerate Three: Autosomal Dominant, Autosomal Recessive Diseases of Genodermatosis ?

- Autosomal Dominant:

- 1- Epidermolysis Bullousa Simplex.
- 3- Neurofibromatosis (NF)

- 2- Ichthyosis Vulgaris
- 4- Tuberous Sclerosis.

- Autosomal Recessive:

- 1- Epidermolysis Bullousa Junctional.
- 2- Lamellar Ichthyosis.
- 3- Xeroderma Pigmentosa (XP).

Q- Define Ichthyosis ?

Group of Disorders which Present at Birth or in Early Childhood.

Characterized by → Chronic, Generalized, Non Inflammatory, Scaling of Skin.

Q- What are the Classification of Ichthyosis ?

- | | |
|-------------------------|-------------------------|
| 1- Ichthyosis Vulgaris. | 2- Lamellar Ichthyosis. |
| 3- X-Linked Ichthyosis | 4- Aquired Ichthyosis. |

Q- What are the Clinical Pictures of Lamellar Ichthyosis ?

Fine Scales or Plate like (Fish Skin), Commonly in Flexure Surface.

- Also Presented with Scarring Alopecia, Ectropion, Bilateral Conjunctivitis, Small Deformed Ears & Inflexible Digits.
- Also Presented with Nail Abnormality which Includes:

- | | |
|---|---|
| 1- Dystrophy with Nail fold Inflammation. | 2- Subungual Hyperkeratosis. |
| 3- Long, Transverse Stippling. | 4- The Nail grow 2-3 Times Faster Than Normal. |

Q- What are the Treatment of Lamellar Ichthyosis ?

- 1- Emollients; (Ex→ Vaseline).
- 2- keratolytic Cream; (10 – 20% Urea Cream).
- 3- May Retinoid Used.

Q- What are the Skin Manifestations of: Neurofibromatosis, Xeroderma Pigmentosa, and Tuberous Sclerosis ?

Neurofibromatosis (<u>CNAL</u>)	Xeroderma Pigmentosa	Tuberous Sclerosis (<u>ASK</u>)
1- <u>Café-au-lait Spots</u> are the <u>Most Characteristic Feature</u> of Disease, as they are Present in 100% Of Cases , (<u>Café-au-lait Spots</u> : are Multiple, Brown, Oval Macules and Patches). 2- <u>Neurofibromas</u> "Skin Tags". 3- <u>Armpit Freckling</u> or Groin. 4- <u>Lisch Nodules</u>. (Also <u>Plexiform Neurofibroma</u>)	1- <u>Photosensitivity</u> → Increase risk of Skin Cancers (BCC, SCC). 2- <u>Pigmented Freckles</u>. 3- <u>Dry & Scaly Skin</u> + Irregular Dark Spots.	1- <u>Ash-leaf Spots</u> (White Patches of Skin Due to lack of Melanine Visible at Birth). 2- <u>Shagreen Patch</u>. 3- <u>Koenen Tumor</u> (Present around & Under Nails of Toes or Fingers). Also: <u>Skin Pigmentation</u> (Café-au-lait Spots).

- VIRAL SKIN INFECTION:

HERPES ZOSTER & POX VIRUS

Q- What is the Causative Organism for Herpes Zoster, & which Type ?

Varicella Zoster, It is DNA Virus.

Q- What are the Criteria for Lesions in Herpes Zoster ?

Multiple Groups of Vesicles on Erythematous Base
Following Dermatomeal Distributions.

Q- How to Diagnose Herpes Zoster ?

Mainly Clinically.

Tzanck Smear → Take Fluid from Vesicle → Dry → Giemza Stain.

Q- What are the Treatment Lines of Herpes Zoster ?

1. Systemic Acyclovir "Zovirax" Orally → 800mg → 5 Times \ Day \ Week.

OR → I.V Acyclovir 5-10mg\ Kg every 8 Hours for Week.

2. Topical Anti-Biotic "Ointment" to **Prevent** Secondary Bacterial infection.

3. Analgesia + **Vit B6,B12**.

Q- Enumerate Five Complication Follow Missed Treatment of Herpes Zoster ?

1- Secondary Bacterial infection.

2- Post Herpatic Neuralgia; (Persistent Sever Pain at Site of lesion, Occurs in **70%** of Pt.

3- Post Herpatic Scar.

4- Ophthalmic Nerve Involvement → Corneal Ulcer & Opacity → Blindness.

5- Facial Nerve Involvement → Facial Nerve Palsy.

6- Sacral Nerves Involvement → Urine Retention.

7- Dissemination of HSV Especially in Immune Suppressant Pt and Children.

Q- Why the Patient Have to Use Treatment for the Eye in Case of Naso-Ciliary Branch Involvement in Addition of Zovirax ?

Because if Naso-Ciliary Nerve Involved Will Leads to;

- 1- Corneal Involvement.
- 2- Acute Retinal Necrosis.

Q- Enumerate Two Side Effect of Zovirax ?

- 1- GIT Upset (Nausea, Vomiting, Diarrhea), 2- Hepato-toxicity, 3- Nephro-toxicity,
- 4- Skin Rash, 5- Pruritis, 6- Urticaria.

Q- What are the Diseases that Caused by Pox Virus ?

- 1- Molluscum Conagiosum
- 2- ORF

WARTS

Q- What are the Types of Warts ?

- | | |
|---|---|
| 1- Common Wart (<u>Varruca Vulgaris</u>). | 2- Plane Wart (<u>Varruca Plana</u>). |
| 3- Planter Wart. | 4- Filiform Wart. |
| 5- Genital Wart. | 6- Mosaic Wart. |

Q- What is the Virus that Cause Wart & is it RNA or DNA ?

Human Papilloma Virus (HPV) & It is DNA Virus.

Q- Enumerate Number of Viruses that Cause Wart ?

- 1- Common Wart → HPV 2 , 4
- 2- Plane Wart → HPV 3
- 3- Planter Wart → HPV 1 , 2 , 4
- 4- Genital Wart → HPV 6 , 11 , 16 , 18

Q- What is the Differential Diagnosis of Planter Warts & How to Differentiate ?

- 1- Callosity.
- 2- Foreign Body.

Planter Wart	Callosity
Present Anywhere in Planter area.	Present Only at Pressure area.
Painful on Side to Side Pressure.	Painful on Vertical Pressure.
Black Dot.	No .
Scraping Lead to Pin-Point Bleeding.	No

Q- What are the Lines of Treatment for Each Type of Wart ?

1. Salicylic acid → Planter Wart.
2. Podophyllin → Genital Wart (C\I in Pregnancy → Teratogenic).
3. Electrodesiccation & Curettage → Plane Wart & Filiform Wart.
4. Cryosurgery → Common Wart (Liquid Nitrogen; -196 C, is the Most Effective Method of Cryosurgery), Liquid Nitrogen is Very Painful if Used in Planter Wart.

Q- If you Suspect Pregnant Patient have Genital Wart, What is your Treatment ?

- 1- Electro-Cautery
- 2- Cryotherapy by Liquid Nitrogen (Especially in Large Wart).

Q- What is the Name of Genital Wart?, Have you Hear Another Name Resemble?, & Where ?

Condyloma Accuminatum.

Yes, in Secondary Stage of Syphilis (Condyloma Lata).

Q- What are the Differential Diagnosis of Koebner Phenomenon ?

- 1- Psoriasis.
- 2- Lichen Planus.
- 3- Vitiligo.
- 4- Wart.
- 5- Molluscum Contagiosum.

- PARASITIC SKIN INFESTATION:

LEISHMANIASIS

Q- What is Reservoir Host & Vector of Leishmaniasis ?

Reservoir Host → Dogs & Rodents.

Vector → Sand Fly (Phlebotomus Paptazi).

Q- What are the Differential Diagnosis of Leishmaniasis ?

- | | |
|--|--------------------------|
| 1- Lupus Vulgaris (<u>TB of Skin</u>). | 2- Furuncle & Carbuncle. |
| 3- Deep Fungal Infection. | 4- Lepromatous Leprosy. |
| 5- Syphilis. | 6- Ecthyma. |
| 7- Malignant Ulcer. | 8- Venous Ulcer. |

Q- What are the Investigations of Leishmaniasis ?

1. Slit & Smear Test: Showing Amastigoite.
2. Culture (NNN Media): Showing Promastigoite.
3. Montenegro Test.

Q- How to Do Slit & Smear Test ?

1. Slit by Knife Until Oozing, and then Take Smear from Dermis.
2. Staining by Giemza Stain.
3. Stage that Seen Under Microscope is Amastigoite.

Q- What is the Stain that Used in Leishmaniasis ?

Giemza Stain.

Q- What is the Stage that be Seen in Leishmaniasis during Staining ?

Amastigoite.

Q- What is the Treatment of Leishmaniasis ?

- May Heals Spontaneously During (6 Months to 1 Year).

1. Pentostam (Sodium Stiboglyconate).

2. Cryotherapy.

3. If No Response; Give → Rifampicin & Isoniazide.

Q- What are the Investigations that have to be Done before Using of Pentostam ?

- Liver Function Test (LFT).

- Renal Function Test (RFT).

- **ECG**

Q- What are the Side Effects of Pentostam ?

1. Hepato-Toxicity.

2. Nephro-Toxicity.

3. **ECG** Changes.

4. Respiratory Irritation.

5. Epigastric Discomfort.

SCABIES & PEDICULOSIS

Q- What are the Lesions of Scabies ?

- Primary Skin Lesion → Burrow

- Secondary Skin Lesion → Scratch Marks (Excoriations), Pustules, and Oozing.

Q- What are the Sites of Scabies ?

Skin Folds; → Axillary Fold, Inter-Digital Fold (**Web**), Under Breast, Umbilicus, Genitalia, Thigh, Gluteal Region, Flexor Aspect of Wrist, Elbow.

In Children; Mostly at Wrist.

In Infant; Mostly in Face, Scalp, Palm, Sole.

Q- What are the Symptoms & Investigations of Scabies ?

- Symptoms of Scabies:

Sever, Persistent, Itching Worse at Night and after Bathing.

- Investigations of Scabies:

- Presence of Burrow at Site of Lesion. - More than One of Family Member involved.
- Microscopic Examination → Scraping from affected area → KOH 10% → Mite, Feces, & Eggs.
- Therapeutic Test → Give Treatment of Scabies; if Pt Respond; → Test is Positive.

Q- What are the Drugs that Used for Treatment of Scabies ?

1. Sulfur Ointment; (2.5% Infant), (5% in Children), (10% in Adult), Following a Hot Bath.
2. Crotamitone; (10%) "Eurax".
3. Malathion; (0.5%)
4. Permethrin; 5% (used in Children).
5. Gamma Benzene Hexachloride; (1%) (CVI in Pregnancy & Children).
6. Benzyl Benzoate.
7. Give Anti-Histamine For Itching.

Q- What are the Types of Scabies ?

1. Infantile Scabies.
2. Adult Scabies.
3. Nodular Scabies.
4. Norwegian Scabies.
5. Animal Scabies.

Q- Does Treat the Family in the Same Time ?

Yes, Because it is Communicable Infection & Spread to Community.

Q- What is the Causative Organism of Scabies ?

Sarcoptes Scabie Mite.

Q- What are the Types of Pediculosis ?

1. Pediculosis Capitis.
2. Pediculosis Corporis.
3. Pediculosis Pubis.

Q- Name Four Drugs Used For Treatment of Pediculosis ?

- **Topical** → Malathion, Benzyl Benzoate, Benzene Hexachlorid.
- **Systemic** → Cotrimoxazole.

BACTERIAL SKIN INFECTION

Q-What are the Skin Diseases that Caused by Staphylococcus & Streptococcus ?

Staphylococcus Infection:	Streptococcus Infection:
1. Impetigo. 2. Ecthyma. 3. Superficial Folliculitis. 4. Deep Folliculitis. 5. Staphylococcal Scalded Skin Syndrome "SSSS"; It is Caused by Bacterial Toxins So it is Indirect Infection.	1. Impetigo. 2. Ecthyma. 3. Cellulitis. 4. Erysipelas. 5. Scarlet Fever; It is Caused by Bacterial Toxins So it is Indirect Infection. 6. Erythema Nodosum.

Q- How to Differentiate Between Cellulitis & Erysipelas ?

	Cellulitis	Erysipelas
Definition	<u>Deep</u> Skin infection in <u>Subcutaneous</u> Tissue.	<u>Superficial</u> Skin infection in <u>Dermis</u> .
Sites	Common in <u>Legs & Feet</u> .	Common in <u>Face</u> .
Clinical Pictures	<u>Dull</u> Red, <u>ill-defined</u> border, Tender, Large area.	<u>Bright</u> Red, <u>Well-defined</u> border Bulla. Also associated with Fever & Malaise.
Cause	Streptococcus, Staphylococcus, H.Influenza in Children .	Caused by → Streptococcus.
Management	1. Complete Bed Rest. 2. Compress Stocks & Elevation of the Leg. 3. Local & Systemic <u>Anti-Biotic</u>. 4. Surgical Debridement.	1. Complete Bed Rest. 2. Compress Stocks & Elevation of the Leg if Lesion was in the Leg. 3. Local & Systemic <u>Anti-Biotic</u>. "Streptococcus → Penicillin"

Q- What is the Causative Organism for Impetigo ?

- Non Bullous Impetigo "Classical": Streptococcus & Staphylococcus.
- Bullous Impetigo: Staphylococcus.

Q- What are the Clinical Types of Impetigo ?

1. Non Bullous Impetigo "Classical".
2. Bullous Impetigo.

Q- What are the Complications of Impetigo ?

1. Abscess.
2. Bacteremia.
3. Toxemia.
- 4 Lymphangitis \ Lymphadenitis.
5. Erysipelas.
6. Cellulitis.
7. Glumerulo-Nephritis; So you can do → Urine analysis & Culture, RFT.

Q- How to Treat Impetigo ?

1. Remove Crust by → Potassium Permanganate.
2. Topical Anti-Biotic → Fusidic acid (Cream or Ointment).
3. In Sever Cases → Systemic Anti-Biotic.

FUNGAL SKIN INFECTION

Q- What are the Types of Tinea ?

Tinea Capitis, Tinea Pedis (Athletic Foot), Tinea Corporis,
Tinea Cruris, Tinea Manuum, Tinea Unguim (Onychomycosis).

Q- Enumerate Types of Tinea Capitis ?

1. Non inflammatory Tinea Capitis: → Patch of Hair loss Causing Non Scaring Alopecia.
2. Inflammatory Tinea Capitis: it has Two Pictures → Kerion & Favus.

Q- How to Differentiate Between Kerion & Favus Type ?

Kerion	Favus
<u>Boggy</u> inflammatory <u>Nodules</u> or Plaques, <u>Very Painful</u> , Draining <u>Pus</u> from <u>Multiple</u> Opening, Hairs <u>Not</u> break but <u>Fallout</u> Easy Leading to <u>Scaring Alopecia</u> .	It is a <u>Chronic</u> infection appear as Thick, <u>Yellow Cup</u> Shaped <u>Crust</u> Named " <u>Scatula</u> " Leading to <u>Scaring Alopecia</u> .

Q- What are the Investigations of Tinea Capitis ?

1. Microscopic with KOH: Collect Sample by Scraping Skin lesion & Put it in Microscopic Slide with KOH 10-20% → If you See Hyphea & Spores; that indicate Fungal Infection.
2. Woods Lamp: Green in Tinea Capitis.

Q- What is the Color of Woods Lamp in Tinea Capitis ?

Green.

Q- What are the Treatment of Tinea ?

1. Tinea pedis, Cruris, Corporis, Mannum → Topical Anti-Fungal → Imadizoles Derivatives in form of Solution, Cream, Ointment. (Ketoconazole, Miconazole, Econazole).

2. Tinea Capitis & Ungium → Systemic Anti-Fungal → **Grisofulvin 20mg \ kg \ Day for 6 Weeks** "Given Orally with Fatty Meal; Because it is Fat Soluble Drug.

S\E of Grisofulvin: GIT Upset, Hepato-toxicity, Headache, Increase risk of SLE, *No Effect against Candidiasis.*

Q- Mention the Lesion that Caused by Pityriasis Versicolor ?

Non Tender, Unilateral or Bilateral, Single or Multiple, Hypopigmented or Hyperpigmented, Macules or Patches Covered with Fine Scale, Mild Itching.

Q- What are the Investigations of Pityriasis Versicolor ?

1. Microscopy with KOH → Hyphea & Spores in appearance Called; **Spaghetti meat form.**
2. Woods Lamp → Golden Yellow.

Q- How to Treat Pityriasis Versicolor ?

1. Systemic Itrakonazole 100mg.
2. **Nizoral** Shampoo as Prophylactic Treatment.
3. Selenium Sulfate Shampoo 2% (SSS).
4. Salicylic acid 2.5%.

Q- What is the Color of Woods Lamp in Pityriasis Versicolor ?

Golden Yellow.

MYCOBACTERIUM SKIN INFECTION

Q- What is the Causative Organism of Leprosy ?

Mycobacterium Leprae.

Q- What are the Types of Leprosy ?

- | | |
|-------------------------------------|--------------------------------------|
| 1. Tuberculoid Leprosy. | 2. Lepromatopus Leprosy. |
| 3. Border Line Tuberculoid Leprosy. | 4. Border Line Lepromatopus Leprosy. |
| 5. Border Line Type. | |

Q- What are the Difference Between Tuberculoid & Lepromatopus Leprosy ?

Tuberculoid Leprosy	Lepromatous Leprosy
- Occur in Patient with Good Immunity→ 90% .	- Occur in Patient with Low Immunity→ 10% .
- Affect <u>Skin & Peripheral Nerves</u> .	- Affect <u>Skin, Mucous Membrane, Internal Organs</u> .
- Has No Systemic Effect.	- Has <u>Systemic</u> Involvement (<u>Bones, Testes, Eyes</u>).
- <u>Less</u> Sever.	- <u>More</u> Sever.
Clinical Picture: - <u>Asymmetrical</u>, Well-Defined, Hypo-Pigmented Macules. - Thickening of Peripheral Nerves. - <u>Loss</u> of Sensation, (1st loss Temperature). - Loss of Sweating (Dry Skin).	Clinical Picture: - <u>Symmetrical</u>, ill-Defined, Hypo-Pigmented or Erythematous Macules. - No Thickening of Peripheral Nerves. - <u>Mild Loss</u> of Sensation. - Normal Sweating.
- Positive Lepromin Test.	- Negative Lepromin Test.

Q- How to Diagnose Leprosy ?

1. Tissue Smear Testing (**Slit & Smear**): Obtain fluid from the lesion, Then The fluid is Placed on Glass Slide and Stained by Using Zeihl-Neelson Stain to look for Organisms.
2. Skin Biopsy.
3. Lepromin Test.

Q- How to Treat Leprosy ?

1. Dapson.
2. Rifampicin, One injection Stop Nasal Discharge.
3. Clofazamine.

Q- What are the Side Effects of the Drug that Used in Treatment of Leprosy ?

S\E of Dapson	S\E of Rifampicin	S\E of Clofazamine
1. Pancytopenia; "Haemolytic Anemia (Especially in pt with G.6.P.D Deficiency) + Leukopenia + Thrombocytopenia".	1. Change in <u>Color</u> of <u>Urine</u> (<u>Orange</u>). 2. Pruritis with or without Rash. 3. GIT Upset & Hepato-toxicity.	1. <u>Brownish</u> Black Skin Discoloration (<u>Bronzing of Skin</u>). 2. Dryness of Skin.
2. Peripheral Neuropathy.	4. Heamolytic <u>Aneamia</u> .	3. Malabsorption.
3. Hepatitis.	5. Thrombocytopenia.	4. Abdominal Pain (<u>Can be Sever</u>).
	6. Drug Drug Intercation (<u>Enzyme Inducer</u>).	

Q- What are the Types of Cutaneous TB ?

1. Lupus Vulgaris.
2. Scrofula.
3. Warty TB (TB Varruca Cutis).

Q- How to Diagnose Cutaneous TB ?

1. Skin Biopsy and Staining by Using Zeihl-Neelson Stain to look for Organisms.
2. Tuberculin Test (Mantoux Test).

Q- How to Treat Cutaneous TB ?

- 1- Lupus Vulgaris & Scrofula → Remove the Scar & Lymph Nodes +
Anti TB Drugs: Isoniazide, Rifampicin, Pyrazinamide, Ethambutol or Streptomycin.
- 2- Warty TB → Surgical Excision.

SEXUAL TRANSMITTED DISEASES

Q- Enumerate the Sexual Transmitted Diseases ?

Bacterial	Viral	Parasitic	Fungal
1. Syphilis; Caused by: Treponema Pallidum. 2. Chancroid; Caused by: Haemophilus Ducreyi. 3. Lympho-Granuloma Venerium; Caused by: Chlamydia Trachomatis. 4. Gonorrhea; Caused by: Neisseria Gonorrhea.	1. Genital Herpes; Caused by Herpes Simplex Virus Type 2. 2. Genital Wart "Comdyloma Accuminata" Caused by: Human Papilloma Virus. 3. AIDS; Caused by: Human Immune Deficiency Virus "HIV". 4. Molluscum Contagiosum; Caused by: Pox Virus.	1. Scabies; Caused by: Sarcoptes Scabie Mite. 2. Pediculosis Pubis; Caused by Pubic Louse. 3. Trichomoniasis; Caused by → Trichomonas Vaginalis.	- Genital Candidiasis; Caused by Candida Albicans.

Q- What are the Skin & Systemic Manifestation of Secondary Syphilis ?

SALMAC :-

Skin Rash, **A**lopecia, **L**ymphadenitis, **M**ucous Patch, **A**rthralgia, **C**ondyloma Lata.

Q- How to Differentiate Between Chancre & Chancroid ?

Chancre	Chancroid
Caused by; Treponema Pallidum.	Caused by; Haemophilus Ducreyi "Gram Negative Bacilli".
Long Incubation Period → 9-90 Days.	Short Incubation Period → 2-5 Days.
Single.	Multiple.
Painless.	Painful.
Hard & Deep.	Soft & Superficial.
Penicillin is Drug Of Choice.	Ciprofloxacin, Erythromycin, Cephalosporin (Penicillin is Not effective).

Q- What are the Investigations that Used for Diagnosis of Syphilis ?

1. Dark Field Examination (DFE): Diagnostic 100% in Primary Syphilis:

“Take Smear, Put it on Slide without Stain, Condense lens and Close Light of Room;
See → Spiral Shape Organisms with “Rotator movement”.

2. TPHA Test (Treponema Pallidum Haemo-Agglutination).

3. VDR: (Venereal Disease Research laboratory).

Q- What are the Treatment of Syphilis ?

-Early Syphilis (Less Than 2 Years):

Single Dose of **Penzetine Penicillin 2.4 Mega unit IM.**

-Late Syphilis (More Than 2 Years):

Penzetine Penicillin 2.4 Mega unit IM \ One Weekly \ for 3 Weeks.

If Patient **Sensitive** to **Penicillin**; Give → **Erythromycin.**

COMMON CASES FOR CLINIC EXAM

SKIN DISORDERS

- 1. Psoriasis**
- 2. Lichen Planus**
- 3. Vitiligo**
- 4. Acne**
- 5. Alopecia**
- 6. Erythema Nodosum**
- 7. Erythema Multiform**
- 8. Ichthyosis**

SKIN INFECTIONS

- 1. Impetigo (Bacterial)**
- 2. Common Wart (Viral)**
- 3. Tinea Capitis (Fungal)**
- 4. Pityriasis Versicolor (Fungal)**
- 5. Leishmaniasis (Parasitic)**